Hypertension, the leading cause of cardiovascular disease and deaths globally, is a well-established, easily detected, and modifiable risk factor for cardiovascular disease. In a recent multinational study, over 40% of the population investigated had elevated blood pressure (BP) and, of those receiving treatment, more than two-thirds did not have their BP controlled. A number of reasons have been identified for this low success rate in controlling hypertension such as an increased susceptibility to hypertension with advancing age, patient non-compliance, physician knowledge deficit, and “clinical inertia,” i.e. when physicians are reluctant to make therapeutic changes. However, especially in instances in which specific treatment decisions are difficult, such as whether or not to begin drug treatment or what drug class to choose, additional information on the source of the hypertension or more details on effects of treatment in a given individual may significantly improve hypertension management.

One issue that is not often considered is the difference between the steady-state and pulsatile components of BP, and how these components are influenced by different physiological factors. The steady-state component, or the mean arterial pressure, is proportional to the product of cardiac output and total peripheral resistance, while the pulse pressure is directly related to arterial stiffness and stroke volume. Most current approaches to the management of hypertension predominantly focus on reducing the steady-state component of BP. This is evident in that the commonly prescribed pharmacological interventions aimed at lowering BP have mechanisms of action that target the reduction of peripheral resistance and/or cardiac output. In patients with isolated systolic hypertension (the most common form of hypertension in those over the age of 60) elevated systolic and pulse pressure is driven by increases in arterial stiffness and stroke volume. Focusing on the reduction of cardiac output and peripheral resistance neglects the pathogenesis of the hypertension.

However, over the last decade the advent of techniques to non-invasively monitor the effects of arterial stiffness (and the accompanying changes in wave reflection) through the analysis of the central BP waveform, or Pulse Wave Analysis (PWA), has provided new and important information relevant to improving BP assessment and management. Information about pressure wave reflections from the peripheral arteries as well as the stiffness of the arteries cannot be obtained through traditional BP. The Augmentation Pressure (AP) and Augmentation Index (AIx), two parameters calculated in PWA, quantify the absolute and relative contribution, respectively, of wave reflection and stiffness to BP. Numerous studies have demonstrated that these parameters are more predictive of incident congestive heart failure, changes in left ventricular mass index, renal function decline, and incident hypertension, among other diseases, than are brachial BPs.

Although no currently available, approved antihypertensive drugs specifically target arterial stiffness, different drug classes affect AP and AIx (and, therefore arterial stiffness and wave reflection) differently. Vasoactive agents such as ACEis, ARBs,
and calcium channel blockers (CCBs) have been shown to significantly improve measures of wave reflection and arterial stiffness while beta-blockers have demonstrated a detrimental effect on these parameters (although the newer vasoactive beta-blockers such as nebivolol do not show this detrimental effect).

In hypertensive patient management, PWA provides information that could offer insight to the pathogenesis of the hypertension. Incorporation of this knowledge into treatment decisions may assist in identifying the intervention likely to offer the most benefit. Furthermore, it would also aid in determining the effectiveness of a particular treatment by determining the extent of the reduction in the effects of arterial stiffness and wave reflections. In other words, PWA allows for a more comprehensive hemodynamic evaluation in the diagnosis, treatment, and management of individuals with hypertension. This was first demonstrated in the BP Guide study when the incorporation of the additional information provided by PWA resulted in less use of medication to achieve BP control.

Consider the 52-year-old diabetic woman who presented in the physician’s office with a brachial BP of 138/72 mmHg, elevated according to the guidelines for diabetics (Fig. 1). Analysis of her central pressure waveform showed that her central pulse pressure and AP were also elevated, indicating a vasoactive agent may be the most effective mode of treatment. After two weeks on an ACEi/CCB combination, the patient’s brachial systolic pressure was significantly lowered to within the guideline recommendations. Additionally, her AP was also reduced to well within the normal range. In this instance, information provided by analysis of the central pressure waveform aided in properly identifying an appropriate therapy that successfully brought the patient’s elevated BP under control.

The current poor success rate of hypertension control leaves much room for improvement. Evaluation of the central blood pressure waveform by PWA is one tool that can aid in the assessment and management of hypertensive individuals; improving the control rate and getting patients to goal more efficiently.

References

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